university of groningen

University Medical Center Groningen

University of Groningen

Arm Motor Control as Predictor for Hypertonia After Stroke

de Jong, Lex D.; Hoonhorst, Maurits H.; Stuive, Ilse; Dijkstra, Pieter U.

Published in:
Archives of Physical Medicine and Rehabilitation

DOI:
10.1016/j.apmr.2011.03.026

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2011

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):
de Jong, L. D., Hoonhorst, M. H., Stuive, I., & Dijkstra, P. U. (2011). Arm Motor Control as Predictor for Hypertonia After Stroke: A Prospective Cohort Study. *Archives of Physical Medicine and Rehabilitation*, *92*(9), 1411-1417. https://doi.org/10.1016/j.apmr.2011.03.026

Copyright
Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the “Taverne” license. More information can be found on the University of Groningen website: https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment.

Take-down policy
If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): http://www.rug.nl/research/portal. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

Download date: 06-05-2023

ORIGINAL ARTICLE

Arm Motor Control as Predictor for Hypertonia After Stroke: A Prospective Cohort Study

Lex D. de Jong, MSc, PT, Maurits H. Hoonhorst, MD, Ilse Stuive, PhD, Pieter U. Dijkstra, PhD

ABSTRACT. de Jong LD, Hoonhorst MH, Stuive I, Dijkstra PU. Arm motor control as predictor for hypertonia after stroke: a prospective cohort study. Arch Phys Med Rehabil 2011;92:1411-7.

Objectives: To analyze the development of hypertonia in the hemiparetic elbow flexors, and to explore the predictive value of arm motor control on hypertonia in a cohort of first-ever stroke survivors in the first 6 months poststroke.

Design: A prospective cohort study.

Setting: A cohort of stroke survivors from a large, university-affilliated hospital in The Netherlands.

Participants: Patients (N=50) with first-time ischemic strokes and initial arm paralysis who were admitted to a stroke unit.

Interventions: Not applicable.

Main Outcome Measures: At 48 hours, 10 to 12 days, 3 and 6 months poststroke, hypertonia and arm motor control were assessed using the Modified Ashworth Scale and the Fugl-Meyer Assessment arm score.

Results: The incidence rate of hypertonia reached its maximum before the third month poststroke (30%). Prevalence was 42% at 3 and 6 months. Participants with poor arm motor control at 48 hours poststroke were 13 times more likely to develop hypertonia in the first 6 months poststroke than those with moderate to good arm motor control. These results were not confounded by the amount of arm function training received.

Conclusions: Hypertonia develops in a large proportion of patients with stroke, predominantly within the first 3 months poststroke. Poor arm motor control is a risk factor for the development of hypertonia.

Key Words: Epidemiology; Muscle hypertonia; Rehabilitation; Risk factors; Stroke; Upper extremity.

© *2011 by the American Congress of Rehabilitation Medicine*

ANNUALLY, 15 MILLION PEOPLE worldwide have a stroke. Five million of them are left permanently disabled, placing a burden on both family and community.[1] In almost 66% of the stroke survivors with initial motor deficits, the affected arm remains without function after 6 months.[2,3] Because of this lack of function, the patient's affected arm remains inactive and immobilized. Over time, the central nervous system and (connective) tissues of the arm adapt to this state of inactivity,[4,5] often resulting in residual impairments such as contracture and hypertonia.

Poststroke hypertonia (increased resistance to passive stretch) has been associated with dependence in everyday activities,[6] motor impairments, activity limitations,[7,8] worse arm motor recovery, and a longer time to admission for rehabilitation.[9] Ideally, knowledge about epidemiologic data concerning hypertonia and its associated prognostic variables might help physiatrists and therapists to recognize how often it occurs and which patients are at greater risk of developing hypertonia so that preventive measures could be taken in daily clinical practice. However, data about the incidence of hypertonia are scarce, and until recently, only a few postacute prognostic variables could be identified as a risk factor 12 months after stroke.[10,11] Besides that, the available data about the prevalence are rather heterogeneous because of differences in patient groups (acute vs chronic/ischemic vs hemorrhagic stroke), assessment timing (ranging between 5.4d and 18mo), assessment methods (Ashworth Scale, Modified Ashworth Scale, Tone Assessment Scale), study design (cross-sectional vs longitudinal), and the clinical definitions of hypertonia.[6,7,9,12,13] Moreover, because hypertonia may fluctuate over time in about 5% to 7.5% of the patients,[7,12,14] data from cross-sectional studies may underestimate or overestimate the prevalence of hypertonia.

The 2 major contributors to hypertonia are reflex hyperexcitability and the passive mechanical properties of the muscle (contracture).[15] A more serious degree of upper motor neuron damage (ie, a larger stroke) results not only in less recovery[16] but also in the development of significantly more clinical manifestations such as reflex hyperexcitability.[17] In addition, full arm paralysis or severe paresis is likely to result in learned nonuse[18] of the hemiplegic arm, increasing the chance of contracture development. Since the combined effects of reflex hyperexcitability and contracture can cause increased resistance to passive stretch, one may expect that patients with stroke who have the most severe brain damage (and hence the

List of Abbreviations

CI	confidence interval
FMA	Fugl-Meyer Assessment
GEE	generalized estimating equations
MAS	Modified Ashworth Scale
OR	odds ratio
OT	occupational therapy
PT	physical therapy
TMS	transcranial magnetic stimulation
t0	prestroke
t1	assessment at 48 hours poststroke
t2	assessment after 10 to 12 days poststroke
t3	assessment at 3 months poststroke
t4	assessment at 6 months poststroke

From De Vogellanden, Center for Rehabilitation, Zwolle (de Jong, Hoonhorst); Department of Rehabilitation Medicine (de Jong, Dijkstra), the Center for Rehabilitation (Stuive), and the Department of Oral and Maxillofacial Surgery (Dijkstra), University Medical Center Groningen, University of Groningen, Groningen, The Netherlands.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit on the authors or on any organization with which the authors are associated.

Reprint requests to Lex D. de Jong, MSc, PT, De Vogellanden, Center for Rehabilitation, Dept of Physiotherapy, PO Box 1057, 8001 BB Zwolle, The Netherlands, e-mail: *l.de.jong@vogellanden.nl.*

0003-9993/11/9209-01084$36.00/0
doi:10.1016/j.apmr.2011.03.026

poorest level of recovery of arm motor control) are more at risk for the development of hypertonia. Although some literature reports on how arm hypertonia evolves poststroke,[12,19] to date little is known about whether hypertonia develops differently in specific subgroups of patients. To test this hypothesis, subgroups have to be formed based on the level of arm motor control. Findings from 2 recent studies showed that patients with severe arm paresis had 10^{19} to 22^{6} times higher odds of having hypertonia at 1 month and 1 year poststroke, respectively, compared with patients with no and moderate paresis. However, in both studies, hypertonia was deemed present if resistance to passive movement was felt during any of 8 different passive arm movements performed, which gives little detail about which muscles are most prone to hypertonia development. Moreover, 1 study[6] was a cross-sectional survey 1 year after stroke, presenting results from which it is difficult to predict who is (most) at risk of developing hypertonia and who may benefit from preventive intervention early after stroke onset. Collecting prospective data from single arm muscle groups would facilitate more accurate predictions.

The aim of this study was 2-fold: (1) to analyze the incidence and prevalence of hypertonia in the hemiplegic elbow flexors during the first 6 months poststroke, and (2) to analyze the influence of motor control and time on the development of hypertonia. We hypothesized that (1) stroke survivors with poor recovery of motor control were more at risk for the development of hypertonia than those with a better level of recovery, and (2) the longer the period after stroke, the greater the risk for hypertonia.

METHODS

Participants and Study Design

The current study was part of a cohort study on the predictive value of transcranial magnetic stimulation (TMS) for recovery in ischemic stroke (M.H. Hoonhorst, unpublished data, 2011). The original cohort, 73 patients with first-ever ischemic strokes who were admitted to a specialized stroke unit of a large university-affiliated hospital (Isala Clinics, Zwolle, The Netherlands), was recruited between August 2005 and February 2008. Eligible participants were those who were unable to elevate the arm while lying in a supine position, unable to voluntarily move the fingers during the first physical screening, or both. After confirmation of the diagnosis by computed tomography or magnetic resonance imaging scan, the patient had to be hospitalized within the first day after the onset of symptoms and had to show a unilateral paralysis or significant paresis of the arm (Medical Research Council score, 0–3). Patients were excluded if they had severe loss of consciousness, were comatose, terminally ill, unable to receive neurorehabilitation because of severe comorbidity, or if contraindications to TMS were present.[20] In addition, patients without clear motor deficits within 24 hours of stroke onset (resulting from a transient ischemic attack) as clinically judged by the neurologist were also excluded. Each participant was assessed within 48 hours (t1), after 10 to 12 days (t2), and after 3 (t3) and 6 (t4) months postonset. All participants or close relatives gave written informed consent. The study was approved by the local medical ethics committee.

Clinical Assessments

At baseline (t1), the participants' characteristics (age, sex, affected side) and Barthel Index scores were collected. Hypertonia was assessed using the 6-point Modified Ashworth Scale (MAS),[21] which is a valid indicator of resistance to passive stretch.[15] During administration of the MAS, the participants were seated in a comfortable position with their forearms in supination. Participants were instructed to relax while the rater first passively moved the forearm from full flexion to full extension to determine the available range of motion. This was done slowly so as not to elicit any reflex activity. Then the same movement was repeated in approximately 1 second by counting "one thousand and one" to rate the actual resistance to passive movement. Clinically relevant hypertonia was operationally defined as an MAS score of at least 1+ (slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder [less than half] of the range of motion). Motor control of the hemiplegic arm was assessed using the 66-point arm section of the Fugl-Meyer Assessment (FMA).[22] The FMA has an excellent reliability, has good construct validity,[23,24] and is highly responsive for changes in motor function after stroke.[25] We considered participants with an FMA score of 18 points or less as having poor motor control, and participants with more than 18 points as having moderate to good motor control. During the t2, t3, and t4 assessments, participants were additionally asked to report their weekly frequency of occupational therapy (OT) and physical therapy (PT), and whether arm function training (yes or no) was part of the treatment program in the weeks before the assessments. All assessments were performed in the hospital or outpatient location by the same rater (M.H.H.).

Statistical Analyses

Descriptive statistics were used to report participant characteristics at baseline. Incidence proportion (number of participants developing hypertonia during a period/total number of participants at risk observed during that period) and prevalence (total number of participants showing hypertonia/total number of participants) were calculated for t1, t2, t3, and t4. At these time points, the frequency of OT and PT and the number of participants receiving arm function training were compared between the 2 arm function groups (poor arm function vs moderate/good arm function) by using an independent t test and a chi-square test, respectively. To estimate the predictive value of poor motor control and days after stroke for the development of hypertonia during the first 6 months, we dichotomized the MAS scores (MAS $\leq$1 and MAS $\geq$1+) and subsequently performed a logistic generalized estimating equations (GEE) analysis by using an exchangeable correlation structure. With logistic GEE we predicted the dichotomous outcome variable (MAS) on the basis of a binary predictor variable (FMA $\leq$18 points at t1, yes or no) and an interval predictor variable (days after stroke), while correcting for dependency of the data within a participant (repeated assessments over time). The interaction between motor control and time was explored whereby time was recoded into days after stroke (t1–t4: 2, 11, 90, and 180d, respectively). For all tests, the 2-tailed significance level was set at .05. All analyses were performed using SPSS (version 16).[a]

RESULTS

Of the initial 73 participants, 22 died and 1 was lost to follow-up (M.H. Hoonhorst, unpublished data, 2011), leaving 50 participants for data analysis. Their baseline characteristics are shown in table 1.

Figure 1 shows how hypertonia (the MAS scores) developed over time. The incidence rate of hypertonia reached its maximum (30%) between t2 (10–12d after stroke) and t3 (3mo after stroke); thereafter the incidence decreased. At 6 months poststroke (t4), 21 participants (42%) had hypertonia (MAS $\geq$1+), of whom 4 (8%) showed a transient course.

Table 1: Characteristics of Participants With a First-Ever Ischemic Stroke at Baseline (N=50)

Characteristics	Values
Age (y)	70.3±12.3
Sex (M/F)	21/29
Paretic side (L/R)	26/24
Barthel Index	5 (2–8.3)

NOTE. Values are mean ± SD, n, or median (interquartile range).
Abbreviations: F, female; L, left; M, male; R, right.

Table 2: Overall Values of FMA Arm Scores and Percentages of Participants With an FMA Score of ≤18 Points and >18 Points at 48 Hours (t1), After 10 to 12 Days (t2), After 3 (t3) and 6 (t4) Months

FMA (N=50)	t1	t2	t3	t4
Median (IQR)	8.5 (3–50.5)	17 (4–58)	51 (5.75–63)	53 (6–64)
≤18 points	60%	52%	38%	36%
>18 points	40%	48%	62%	64%

Abbreviation: IQR, interquartile range.

Table 2 shows that 40% of the participants already had more than 18 points on the FMA at t1. This percentage increased to 64% at t4. At 6 months postonset, 36% of the participants had 18 points or less on the FMA, and their median FMA scores never exceeded a total of 5 points. In the other subgroup, recovery of arm motor control seemed to have stabilized at about 3 months poststroke with a median of 61 points.

Between the 2 FMA subgroups, no significant differences (*P* values ranging from .074 to .98) with regard to the frequencies of OT and PT were found for any of the time points (table 3). The total frequencies per group were also not significantly different (calculations not shown). Table 3 further shows that only 0% to 4% of the participants with more than 18 points on the FMA received arm training from physical therapists. Occupational therapists continued arm treatment in most participants irrespective of the level of arm function until t3. After t3, only 13% of the participants with 18 points or less on the FMA continued receiving arm training from occupational therapists.

Results from the GEE (table 4) revealed that an FMA score of 18 points or less was a significant predictor for the presence of hypertonia; these participants were 12.8 (95% confidence interval [CI], 3.5–47.3) times more likely to develop hypertonia (MAS ≥1+). Days after stroke (time) also was a significant predictor of hypertonia; per day beta increased with .011. The interaction between motor control and time was not significant (*P*=.58).

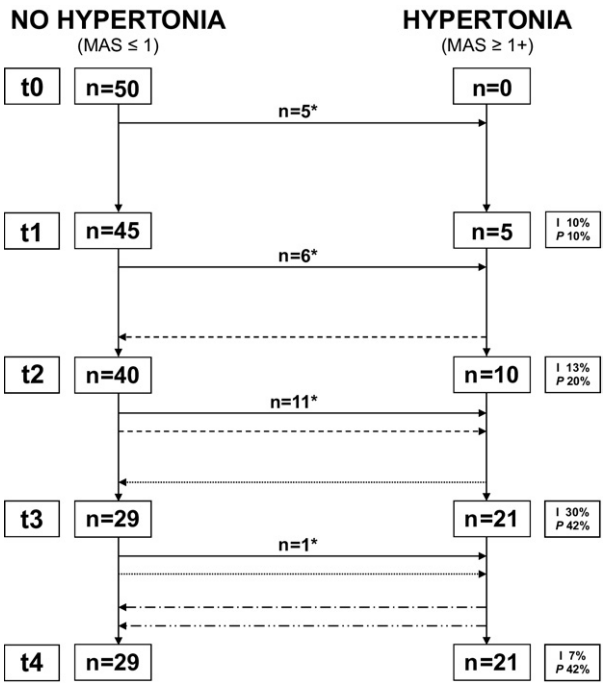

Fig 1. Flow chart depicting the development of hypertonia (MAS ≥1+), incidence proportions, and prevalence rates in/of 50 subjects during the first 6 months poststroke. Solid arrows represent participants who developed permanent hypertonia. Dotted arrows represent the 4 individuals who showed a transient course of hypertonia. Abbreviations: I, incidence proportion; P, prevalence rate; t0, prestroke; t1, at 48 hours; t2, at 10–12 days; t3, at 3 months; t4, at 6 months. Asterisk, number of participants who develop hypertonia for the first time.

DISCUSSION

To our knowledge, this is the first longitudinal study describing in detail both the incidence and the prevalence of elbow flexor hypertonia in the first 6 months poststroke, as well as the predictive value of arm motor control on its development. The incidence rate of hypertonia reached its maximum before the third month poststroke (30%). A large portion (42%) of the participants had hypertonia at 3 and 6 months postonset. The present study also shows that participants with poor motor control (ie, ≤18 points on the FMA) at 48 hours poststroke were 13 times more likely to develop hypertonia in the first 6 months poststroke than those with FMA scores more than 18 points. Arm function training did not confound these findings. Additionally, the risk of developing hypertonia increased significantly over time.

Study Limitations

Some limitations of this study have to be addressed. First, only elbow flexor hypertonia was assessed with the commonly used MAS. Although there is considerable debate about the clinimetric properties of the MAS, several studies[26-28] have shown that its reliability is sufficiently high (eg, weighted kappa=.84 for interrater and .83 for intrarater comparisons[27]) when used to assess hypertonia in the elbow joint. However, because elbow flexors are not the only arm muscles prone to the development of hypertonia, the prevalence and incidence of hypertonia of the affected arm after stroke may have been underestimated in the current study. Second, because we dichotomized the MAS scores, comparisons with the findings of other investigators may be hindered. We argue that clinically, MAS scores of 1 or less are not relevant, and patients with these scores do not receive interventions aimed at decreasing hypertonia. Further, in our opinion, the "catch and release" phenomenon as part of score 1 of the MAS is caused by a level of reflex hyperexcitability incapable of causing contracture. In addition, to be able to distinguish between no and clinically relevant hypertonia, the rater has to be able to detect clearly the differences in resistance to movement. When hypertonia was quantified biomechanically, subjects with an MAS of 1+ proved to have significantly higher resistance to passive movements than subjects with an MAS of 0 or 1,[29] which may be a prerequisite to detect these differences. However, in later re-

Table 3: Between-Group Comparison of Possible Confounders of Arm Hypertonia Between 48 Hours and 6 Months Poststroke

Variables	t1–t2		t2–t3		t3–t4	
	FMA ≤18	FMA >18	FMA ≤18	FMA >18	FMA ≤18	FMA >18
PT frequency (sessions/wk)	3.25±0.53 (n=24)	3.36±0.58 (n=22)	2.89±1.05 (n=19)	3.07±1.08 (n=30)	2.11±0.96 (n=18)	2.10±1.35 (n=31)
Participants receiving arm function training by physical therapist (%)	40* (n=20)	0* (n=16)	53* (n=15)	4* (n=24)	62* (n=13)	4* (n=24)
OT frequency (sessions/wk)	2.62±0.65 (n=24)	2.95±0.58 (n=22)	2.33±1.09 (n=18)	2.54±1.14 (n=26)	1.28±1.07 (n=18)	1.27±1.46 (n=30)
Participants receiving arm function training by occupational therapist (%)	100 (n=21)	100 (n=17)	87 (n=15)	96 (n=22)	13* (n=8)	73* (n=15)

NOTE. Values are mean ± SD or as otherwise indicated. n does not add up to 50 because of missing data.
*Significantly (χ^2, Fisher exact test $P<.05$) less OT from t3–t4 and more PT at all occasions for participants with poor motor control (FMA ≤18 points).

Table 4: Prediction of Hypertonia (MAS ≥1+) in the First 6 Months Poststroke

Predictors	β	SE	P	OR (95% CI)
Poor arm motor control at t1	2.55	.67	<.001	12.78 (3.46–47.25)
Days after stroke	0.011	.004	.003	1.01* (1.00–1.02)
Constant	–3.17	.56	<.001	0.04 (0.01–0.13)

NOTE. Results from GEE (N=50).
Abbreviation: OR, odds ratio.
*It may seem that the effects of time can be neglected since the OR is 1.01 per day, but the influence of, for example, 30 days is considerable, resulting in an OR of $1.4 = e^{(30* .011)}$.

search, differences in resistance between grades 1, 1+, and 2 could not be confirmed.[30] Future research is warranted to solve this issue. Third, only data of surviving participants were included in the analysis because they completed all assessments needed for the prediction model. Probably these participants had less extensive strokes than those who died in the course of the study. This selection probably resulted in an underestimation of incidence and prevalence data. Finally, the amount of arm function training participants received from occupational and physical therapists during their participation did not confound the outcomes under study, but because some data were missing, the influence of arm function training needs further investigation in future studies.

In our study sample, the incidence rate of elbow flexor hypertonia was low at 48 hours and at 10 to 12 days (10%–13%), and reached a 30% maximum at 3 months poststroke with an additional 7% after 6 months. The prevalence steadily increased from 10% to 20% in the first 10 to 12 days to a maximum of 42% at 3 and 6 months poststroke. Comparison of these results with those of others (who claimed to have assessed spasticity, but who used the MAS and thus assessed hypertonia[30]) is hampered by differences in methodology. In a frequently cited prospective study by Sommerfeld et al[7] (n=95), an incidence rate of 21% was found at 5.4 days after stroke. Three months after stroke an incidence rate of 3% was found. The prevalence was 19% at that time. Their higher initial incidence rate could be explained by the cutoff point applied (MAS >0) and because the assessment was not limited to the elbow joint only. However, if this argument were true, a higher incidence and prevalence would also be expected at 3 months poststroke in that study. More recently, a 4% incidence rate of hypertonia (MAS ≥1) at 2 to 10 days poststroke was reported. The prevalence was 23% at 6 months poststroke.[19] Since the cutoff point for hypertonia was lower than in our study, the differences between our studies cannot be explained adequately. Yet another definition of hypertonia (Ashworth Scale ≥2) was applied in a study[12] where hypertonia in the elbow and wrist was observed at time points similar to those of our study. The overall incidence rate in that study was very high: 63%. This high rate probably occurred because the most severely affected patients (suffering from first-ever or previous strokes) were selected in order to increase the chance of identifying risk factors for early or persistent hypertonia. Although no correlation was found between "early" hypertonia and previous stroke in that study, confounding (hypertonia from a previous stroke) may have affected the results. We therefore argue that it is best to only select patients with first-ever strokes.

The diversity in patient selection, joint assessment, moments of evaluation, and cutoff points for hypertonia illustrates the difficulty of comparing the different research results. Therefore

a general overview of how many patients with stroke develop hypertonia can still not be given. What does seem clear, however, is that hypertonia develops predominantly within the first 3 months poststroke[12,14] and that it has a transient course in a small subgroup of patients.[7,12,14,19] We also observed these 2 patterns in our sample, resulting in a maximum incidence rate and prevalence of 30% and 42%, respectively, and a transient course of hypertonia in 8% of our participants. This latter feature of hypertonia stresses the need for studying not only prevalences but also incidence rates by means of longitudinal study designs from the acute phase on.

To be able to analyze the influence of the level of motor control on the development of hypertonia, we divided our participants into 2 distinct FMA groups. We used an 18-point cutoff score because poststroke recovery trends indicate that those who have the poorest level of recovery score around 18 points in the chronic phase poststroke,[31,32] and a score of less than 19 points within 4 weeks poststroke is a strong indicator for poor outcome at 6 months.[33] By applying this cutoff score we managed to make a clear distinction between those with poor arm motor control (typically showing only hyperreflexia and mass synergy patterns of shoulder internal rotation, finger and elbow flexion) and those with moderate to good motor control. In the current study, the poor recovery group represented 60% of the 50 participants at 48 hours and 36% at 6 months poststroke. During their participation, all participants received a comparable amount of OT and PT. Participants with the poorest level of arm motor control kept receiving arm PT up to 6 months poststroke. A comparable number of participants in both arm function groups received OT, which was discontinued in the poor function group only after arm motor control had stabilized at 3 months poststroke. This led us to conclude that arm training did not confound hypertonia development. However, one could instead argue that hypertonia was aggravated by the arm function training, a hypothesis that would require further investigation.

Results from our analysis showed that time was a significant contributor to hypertonia development. This was not surprising because both reflex hyperexcitability (resulting from reorganization within the central nervous system) and contracture (resulting from secondary soft tissue changes) need time to develop.[17] Although the resulting OR suggests that the effect of time could be neglected (1.01 per day), the influence of, for example, 30 days is considerable, resulting in an OR of $1.4 = e^{(30 * 0.011)}$. Participants with poor arm motor control 48 hours poststroke had a 13 (95% CI, 3.5–47.3) times higher OR of developing hypertonia in the subsequent 6 months compared with those with moderate to good motor control. This result is in concordance with an OR of 10 (95% CI, 2.1–48.4) found in a nearly similar sample,[19] despite another definition of hypertonia (MAS $\geq$1 in any of 4 arm joints). In another study,[6] an OR of 22 (95% CI, 3.9–125) was found in a subgroup of participants with the most severe paresis. Despite our smaller sample size, our 95% CIs for the estimated ORs were smaller. Maybe our study sample was more homogenous with respect to the level of arm motor control. However, this assumption cannot be verified because of differences in arm motor control assessment. To analyze the effect of the cutoff point for the FMA, we performed a post hoc sensitivity analysis. The resulting ORs for poor motor control were 14.5 and 9.3 when using an FMA cutoff point of 9 and 36 points, respectively. These results indicate that the level of the cutoff point has an influence on the outcome, but they also show that the FMA is a fairly robust predictor of hypertonia development.

Our findings have shown that elbow flexor hypertonia develops in a considerable subgroup of patients with stroke. Our findings also imply that as early as 48 hours poststroke, one can predict who is most at risk for hypertonia development based on the FMA score. This knowledge might serve physiatrists and therapists to inform their patients about the risk of developing hypertonia. It might also serve therapists to take appropriate preventive actions (although a tailored treatment would require more detailed discrimination between the neural and tissue-related components of hypertonia). Despite these findings, hypertonia also developed in a number of participants with moderate to good arm function, which underscores the need for awareness of this impairment in all patients with stroke.

As mentioned earlier, 2 of the main factors that contribute to hypertonia are reflex hyperexcitability and the passive mechanical properties of the muscle (contracture).[15] Reflex hyperexcitability may be hard to influence in the early stages after stroke. Physical and occupational therapists can, however, prevent contracture development in an attempt to keep down the level of resistance to passive movement. Patients with stroke who have an FMA score of 18 points or less could, for example, have measures implemented to prevent contractures. Such measures are currently not instituted soon enough after stroke.[34] Future prospective research should be performed to assess whether such measures, initiated in the acute phase poststroke, can help to reduce both the incidence and the prevalence of hypertonia and its associated features. To obtain a valid overall picture of the development of hypertonia, it seems paramount to perform more longitudinal research, to only select patients with first-ever strokes, to assess single joints separately, and to use similar definitions for hypertonia. In addition, it may be valuable to simultaneously assess clinical variables that seem to influence or predict the development of hypertonia, and to present raw data (Appendix 1) that enables better comparisons between studies. Finally, we urge future researchers to abandon the use of the word "spasticity" when, in fact, hypertonia (resistance to passive stretch) is assessed by using the MAS. This also implies reevaluation of epidemiologic data concerning spasticity as, for example, defined by Lance.[35]

Acknowledgments: We thank J.S.P. van den Berg, MD, PhD, from the Department of Neurology and C.H. Emmelot, MD, PhD, from the Department of Rehabilitation Medicine of the Isala Klinieken in Zwolle for their active recruitment of participants.

APPENDIX 1: PATIENT RAW FUGL-MEYER ASSESSMENT AND MODIFIED ASHWORTH SCALE DATA AT 48 HOURS (t1), 10 TO 12 DAYS (t2), 3 (t3) AND 6 MONTHS (t4) POSTSTROKE

	t1		t2		t3		t4	
Participant	FMA	MAS	FMA	MAS	FMA	MAS	FMA	MAS
01	6	0	6	1	40	2	41	2
02	21	0	17	0	59	0	55	1
03	59	0	58	0	65	0	57	0
04	57	0	66	0	63	0	66	0
05	11	0	59	0	66	0	63	0
06	63	0	66	0	66	0	66	0
07	62	0	65	0	66	0	65	0
08	66	0	66	0	66	0	66	0
09	52	0	58	0	51	0	49	0
10	65	0	66	0	66	0	66	0
11	5	1	6	0	6	1	5	1
12	35	0	55	0	55	0	65	0
13	59	0	61	0	63	0	66	0
14	3	1	32	1	27	1	52	1
15	52	0	63	0	64	0	65	0
16	64	0	64	0	64	0	64	0
17	0	0	0	0	6	0	12	0
18	8	0	9	0	6	0	7	0
19	12	1+	17	1	34	3	38	3
20	5	0	3	0	2	0	1	1
21	3	0	4	0	6	2	8	2
22	5	0	58	0	62	2	64	2
23	2	0	20	1+	58	2	59	2
24	61	0	61	0	66	0	66	0
25	2	0	4	0	3	1+	3	2
26	63	0	65	0	65	1+	65	1
27	4	0	4	1+	10	2	9	2
28	4	1+	6	1+	4	2	6	3
29	9	0	7	1	4	0	4	0
30	2	0	6	1	6	1+	6	1+
31	12	0	12	0	60	0	62	0
32	5	1+	31	1+	36	2	27	2
33	5	1	32	1	61	1	61	1
34	38	1	53	1	65	0	65	0
35	7	0	7	1+	15	1	22	2
36	2	0	7	0	4	2	2	2
37	39	0	34	0	61	0	59	0
38	3	0	2	1	4	0	4	0
39	0	0	0	0	2	0	6	0
40	26	0	2	0	62	0	61	0
41	0	1	21	2	58	3	64	3
42	4	2	4	2	5	2	5	1
43	50	0	10	0	51	1+	54	2
44	46	0	1	1	4	1+	2	2
45	2	0	2	3	2	2	2	2
46	2	0	2	1+	2	2	2	2
47	3	2	15	3	24	1+	25	2
48	18	0	57	0	60	0	60	0
49	1	0	1	0	5	1	6	1+
50	42	0	56	0	59	0	59	0
	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)
	8.5 (3–50.5)	0 (0–0)	17 (4–58)	0 (0–1)	51 (5.75–63)	0 (0–3)	53 (6–64)	1 (0–3)

NOTE. The FMA arm score ranges from 0 to 66.
Abbreviation: IQR, interquartile range.

References

1. Mackay J, Mensah G. The atlas of heart disease and stroke. Geneva: World Health Organization; 2004.
2. Sunderland A, Tinson D, Bradley L, Hewer RL. Arm function after stroke. An evaluation of grip strength as a measure of recovery and a prognostic indicator. J Neurol Neurosurg Psychiatry 1989;52:1267-72.
3. Wade DT, Langton-Hewer R, Wood VA, Skilbeck CE, Ismail HM. The hemiplegic arm after stroke: measurement and recovery. J Neurol Neurosurg Psychiatry 1983;46:521-4.
4. Pandyan AD, Cameron M, Powell J, Stott DJ, Granat MH. Contractures in the post-stroke wrist: a pilot study of its time course of development and its association with upper limb recovery. Clin Rehabil 2003;17:88-95.
5. Lincoln NB, Parry RH, Vass CD. Randomized, controlled trial to evaluate increased intensity of physiotherapy treatment of arm function after stroke. Stroke 1999;30:573-9.
6. Lundström E, Terent A, Borg J. Prevalence of disabling spasticity 1 year after first-ever stroke. Eur J Neurol 2008;15: 533-9.
7. Sommerfeld DK, Eek EU, Svensson AK, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. Stroke 2004;35: 134-9.
8. Katz RT, Rovai GP, Brait C, Rymer WZ. Objective quantification of spastic hypertonia: correlation with clinical findings. Arch Phys Med Rehabil 1992;73:339-47.
9. Dajpratham P, Kuptniratsaikul V, Kovindha A, Kuptniratsaikul PS, Dejnuntarat K. Prevalence and management of poststroke spasticity in Thai stroke patients: a multicenter study. J Med Assoc Thai 2009;92:1354-60.
10. Urban PP, Wolf T, Uebele M, et al. Occurrence and clinical predictors of spasticity after ischemic stroke. Stroke 2010;41: 2016-20.
11. Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK, Watkins CL. Predicting spasticity after stroke in those surviving to 12 months. Clin Rehabil 2004;18:438-43.
12. van Kuijk AA, Hendricks HT, Pasman JW, Kremer BH, Geurts AC. Are clinical characteristics associated with upper-extremity hypertonia in severe ischaemic supratentorial stroke? J Rehabil Med 2007;39:33-7.
13. Watkins CL, Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK. Prevalence of spasticity post stroke. Clin Rehabil 2002;16:515-22.
14. Welmer AK, von Arbin M, Widen Holmqvist L, Sommerfeld DK. Spasticity and its association with functioning and health-related quality of life 18 months after stroke. Cerebrovasc Dis 2006;21: 247-53.
15. Johnson G. Measurement of spasticity. In: Barnes M, Johnson G, editors. Upper motor neurone syndrome and spasticity. Cambridge: Cambridge University Pr; 2001. p 79-95.
16. Binkofski F, Seitz RJ, Hacklander T, Pawelec D, Mau J, Freund HJ. Recovery of motor functions following hemiparetic stroke: a clinical and magnetic resonance-morphometric study. Cerebrovasc Dis 2001;11:273-81.
17. Sheean G. Neurophysiology of spasticity. In: Barnes M, Johnson G, editors. Upper motor neurone syndrome and spasticity. Cambridge: Cambridge University Pr; 2001. p 12-78.
18. Uswatte G, Taub E. Constraint-induced movement therapy: new approaches to outcome measurement in rehabilitation. In: Struss D, Winocur G, Robertson I, editors. Cognitive neurorehabilitation: a comprehensive approach. Cambridge: Cambridge University Pr; 1999. p 215-29.
19. Lundström E, Smits A, Terent A, Borg J. Time-course and determinants of spasticity during the first six months following first-ever stroke. J Rehabil Med 2010;42:296-301.
20. Wassermann EM. Risk and safety of repetitive transcranial magnetic stimulation: report and suggested guidelines from the International Workshop on the Safety of Repetitive Transcranial Magnetic Stimulation, June 5–7, 1996. Electroencephalogr Clin Neurophysiol 1998;108:1-16.
21. Bohannon RW, Smith MB. Interrater reliability of a Modified Ashworth Scale of muscle spasticity. Phys Ther 1987;67:206-7.
22. Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S. The post-stroke hemiplegic patient. 1. A method for evaluation of physical performance. Scand J Rehabil Med 1975;7:13-31.
23. Croarkin E, Danoff J, Barnes C. Evidence-based rating of upper-extremity motor function tests used for people following a stroke. Phys Ther 2004;84:62-74.
24. Gladstone DJ, Danells CJ, Black SE. The Fugl-Meyer Assessment of Motor Recovery After Stroke: a critical review of its measurement properties. Neurorehabil Neural Repair 2002;16:232-40.
25. Hsieh YW, Wu CY, Lin KC, Chang YF, Chen CL, Liu JS. Responsiveness and validity of three outcome measures of motor function after stroke rehabilitation. Stroke 2009;40:1386-91.
26. Gregson JM, Leathley MJ, Moore AP, Smith TL, Sharma AK, Watkins CL. Reliability of measurements of muscle tone and muscle power in stroke patients. Age Ageing 2000;29:223-8.
27. Gregson JM, Leathley M, Moore AP, Sharma AK, Smith TL, Watkins CL. Reliability of the Tone Assessment Scale and the Modified Ashworth Scale as clinical tools for assessing poststroke spasticity. Arch Phys Med Rehabil 1999;80:1013-6.
28. Pandyan AD, Johnson GR, Price CI, Curless RH, Barnes MP, Rodgers H. A review of the properties and limitations of the Ashworth and Modified Ashworth Scales as measures of spasticity. Clin Rehabil 1999;13:373-83.
29. Pandyan AD, Price CI, Rodgers H, Barnes MP, Johnson GR. Biomechanical examination of a commonly used measure of spasticity. Clin Biomech (Bristol, Avon) 2001;16:859-65.
30. Pandyan AD, Price CI, Barnes MP, Johnson GR. A biomechanical investigation into the validity of the Modified Ashworth Scale as a measure of elbow spasticity. Clin Rehabil 2003;17:290-3.
31. Mirbagheri MM, Rymer WZ. Time-course of changes in arm impairment after stroke: variables predicting motor recovery over 12 months. Arch Phys Med Rehabil 2008;89:1507-13.
32. Duncan PW, Lai SM, Keighley J. Defining post-stroke recovery: implications for design and interpretation of drug trials. Neuropharmacology 2000;39:835-41.
33. Kwakkel G, Kollen BJ, van der Grond J, Prevo AJ. Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. Stroke 2003;34: 2181-6.
34. Borisova Y, Bohannon RW. Positioning to prevent or reduce shoulder range of motion impairments after stroke: a meta-analysis. Clin Rehabil 2009;23:681-6.
35. Lance J. Symposium synopsis. In: Feldman R, Young R, Koella W, editors. Spasticity: disordered motor control. Chicago: Chicago Yearbook Medical Publications; 1980. p 485-94.

Supplier

a. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.